```
=> d his

     (FILE 'HOME' ENTERED AT 08:47:58 ON 21 MAR 2005)

     FILE 'REGISTRY' ENTERED AT 08:48:06 ON 21 MAR 2005
                E BOTULIN A/CN
L1              1 S E3
                E BOTULIN B/CN
L2              1 S E3
                E BOTULIN C/CN
L3              1 S E3
                E BOTULIN D/CN
L4              1 S E3
                E BOTULIN E/CN
L5              1 S E3
                E BOTULIN F/CN
L6              1 S E3
                E BOTULIN G/CN
L7              1 S E3
L8              7 S L1-L7

     FILE 'HCAPLUS' ENTERED AT 08:49:16 ON 21 MAR 2005
L9           1141 S L8
                E MAMMARY GLAND /CT
                E E3+A
                E MAMMARY GLAND /CT
                E E3+AL
                E E3+ALL
                E E9+ALL
L10         48363 S MAMMARY GLAND, DISEASE+NT/CT
L11             5 S L9 AND L10
L12          1591 S BREAST (L) (DISEAS? OR DISORDER#)
L13             0 S L12 AND L9
L14          4184 S L9 OR BOTULIN OR BOTULINUM
L16           791 S SNAP 25
L17             1 S L16 AND L10

     FILE 'WPIDS' ENTERED AT 08:55:59 ON 21 MAR 2005
L18           518 S BOTULIN OR BOTULINUM
L19         21014 S (BREAST OR MAMMARY)

     FILE 'MEDLINE' ENTERED AT 08:58:38 ON 21 MAR 2005
                E BOTULIN/CT
                E E3+ALL
                E E2+ALL
L21          5863 S BOTULINUM TOXINS+NT/CT
                E MAMMARY GLAND DIS/CT
L22            46 S MAMMARY GLAND DIS?
                E BREAST DISEASES/CT
                E E3+NT/CT
L23        129296 S BREAST DISEASES+NT/CT

     FILE 'BIOSIS' ENTERED AT 09:03:48 ON 21 MAR 2005
L25          7698 S BOTULIN OR BOTULINUM
L26        194432 S (BREAST OR MAMMARY)
L27             6 S L25 (S) L26
L28            22 S L25 AND L26
L29        144806 S L26 (S) (CANCER OR TUMOR OR NEOPLAS? OR CARCINOM? OR HYPERPLA

     FILE 'EMBASE' ENTERED AT 09:07:36 ON 21 MAR 2005
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                E MAMMARYG GLAND/CT
                E MAMMARY GLAND/CT
                E E12+ALL
                E E2+ALL
                E E1+BT/CT
                E E1+ALL
                E BREAST DISEAS/CT
                E E4+ALL
L31        132759 S BREAST DISEASE+NT/CT
L32          8800 S BOTULIN OR BOTULINUM
L33            30 S L31 AND L32
                E BOTULINUM TOXIN/CT
                E BOTULINUM TOXIN/CT
                E E3+ALL
L34          3952 S BOTULINUM TOXIN+NT/CT

     FILE 'MEDLINE' ENTERED AT 09:11:58 ON 21 MAR 2005

     FILE 'MEDLINE, EMBASE, BIOSIS, WPIDS, HCAPLUS' ENTERED AT 09:12:38 ON 21
     MAR 2005
L37            42 DUP REM L36 L35 L30 L20 L15 (13 DUPLICATES REMOVED)
                E BRIN M/AU
L38           691 S E3 OR E5 OR E11-13
                E DONOVAN S/AU
L39           628 S E3-16
                E DONOVAN STEVEN/AU
                E DONOVAN STEVEN/AU
                E DONOVAN STE/AU
L40           206 S E4-12
L41          1559 S L37-L40
L42         29516 S BOTULIN OR BOTULINUM
L43           309 S L41 AND L42
L44            41 S L43 AND (BREAST OR MAMMARY)
L45            38 DUP REM L44 (3 DUPLICATES REMOVED)
L46             0 S L45 NOT L37
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< inventors, all hits in L37

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=> fil medline embase biosis wpids hcaplus
FILE 'MEDLINE' ENTERED AT 09:17:51 ON 21 MAR 2005

FILE 'EMBASE' ENTERED AT 09:17:51 ON 21 MAR 2005
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FILE 'BIOSIS' ENTERED AT 09:17:51 ON 21 MAR 2005
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FILE 'WPIDS' ENTERED AT 09:17:51 ON 21 MAR 2005
COPYRIGHT (C) 2005 THE THOMSON CORPORATION

FILE 'HCAPLUS' ENTERED AT 09:17:51 ON 21 MAR 2005
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PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2005 AMERICAN CHEMICAL SOCIETY (ACS)

=> d bib ab l37 1-42
THE ESTIMATED COST FOR THIS REQUEST IS 107.72 U.S. DOLLARS
DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N:y

L37  ANSWER 1 OF 42  WPIDS  COPYRIGHT 2005 THE THOMSON CORP on STN DUPLICATE 1
AN   2005-131969 [14]    WPIDS
CR   2001-006327 [01]; 2002-179993 [23]; 2002-254424 [30]; 2002-453014 [48];
     2002-673634 [72]
DNC  C2005-043384
TI   Use of a botulinum neurotoxin to treat cancers of e.g.
     mammary gland, central nervous system, blood cell, colon, rectum,
     skin and prostate.
DC   B04
IN   BRIN, M F; DONOVAN, S
PA   (ALLR) ALLERGAN INC
CYC  1
PI   US 2005031648   A1 20050210 (200514)*      34
ADT  US 2005031648 A1 CIP of US 1999-454842 19991207, CIP of US 2000-631221
     20000802, CIP of US 2002-71826 20020208, US 2004-929040 20040827
FDT  US 2005031648 A1 CIP of US 6139845
PRAI US 2004-929040       20040827; US 1999-454842       19991207;
     US 2000-631221       20000802; US 2002-71826        20020208
AB   US2005031648 A UPAB: 20050228
     NOVELTY - Treatment of a cancer comprises administration of a botulinum
     neurotoxin (I).
          ACTIVITY - Cytostatic.
          MECHANISM OF ACTION - None given.
          USE - (I) is useful in the treatment of cancer, mammary
     gland cancer (breast ductal carcinoma), central nervous system
     cancer (neuroblastoma), blood cell cancer (leukemia), colon cancer, rectum
     cancer, skin cancer (melanoma) and prostate cancer (claimed). The ability
     of (I) to inhibit breast ductal cancer cells (ZR-75) was tested
     in vitro. The results showed that the percentage inhibition by
     botulinum toxins type A (0.1 U/ml) was 28.
          ADVANTAGE - The botulinum toxin type A has more potent and/or longer
     duration of activity. (I) is without any significant deleterious effect.
     Dwg.0/10

L37  ANSWER 2 OF 42  EMBASE  COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
     on STN
AN   2005098013  EMBASE
TI   Antibody Engineering - IBC's 15th Annual International Conference. 30
```

November - 3 December 2004, San Diego, CA, USA.
AU Haurum J.S.
CS J.S. Haurum, Symphogen A/S, Elektrovej, Building 375, DK-2800 Lyngby, Denmark. jh@symphogen.com
SO IDrugs, (2005) 8/2 (91-93).
ISSN: 1369-7056 CODEN: IDRUFN
CY United Kingdom
DT Journal; Conference Article
FS 026 Immunology, Serology and Transplantation
027 Biophysics, Bioengineering and Medical Instrumentation
029 Clinical Biochemistry
030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LA English

L37 ANSWER 3 OF 42 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 2005037156 EMBASE
TI The vulvodynia guideline.
AU Haefner H.K.; Collins M.E.; Davis G.D.; Edwards L.; Foster D.C.; Hartmann E.H.; Kaufman R.H.; Lynch P.J.; Margesson L.J.; Moyal-Barracco M.; Piper C.K.; Reed B.D.; Stewart E.G.; Wilkinson E.J.
CS Dr. H.K. Haefner, Univ. of MI Ctr. for Vulvar Diseases, University of Michigan Hospitals, L4000 Women's Hospital, 1500 East Medical Center Drive, Ann Arbor, MI 48109, United States. haefner@umich.edu
SO Journal of Lower Genital Tract Disease, (2005) 9/1 (40-51).
Refs: 48
ISSN: 1089-2591 CODEN: JLGDFI
CY United States
DT Journal; Conference Article
FS 008 Neurology and Neurosurgery
010 Obstetrics and Gynecology
037 Drug Literature Index
038 Adverse Reactions Titles
039 Pharmacy
LA English
SL English
AB Objective. To provide a review of the literature and make known expert opinion regarding the treatment of vulvodynia. Materials and Methods. Experts reviewed the existing literature to provide new definitions for vulvar pain and to describe treatments for this condition. Results. Vulvodynia has been redefined by the International Society for the Study of Vulvovaginal Disease as vulvar discomfort in the absence of gross anatomic or neurologic findings. Classification is based further on whether the pain is generalized or localized and whether it is provoked, unprovoked, or both. Treatments described include general vulvar care, topical medications, oral medications, injectables, biofeedback and physical therapy, dietary changes with supplementations, acupuncture, hypnotherapy, and surgery. No one treatment is clearly the best for an individual patient. Conclusions. Vulvodynia has many possible treatments, but very few controlled trials have been performed to verify efficacy of these treatments. Provided are guidelines based largely on expert opinion to assist the patient and practitioner in dealing with this condition.

L37 ANSWER 4 OF 42 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN DUPLICATE 2
AN 2004-239192 [22] WPIDS
DNC C2004-093674
TI Generating a population of dendritic cells, useful for inducing protective immune response against infections, cancers or autoimmune diseases by

culturing or expanding CD34+ precursor cells in the presence of one or more cytokines.

DC B04 D16
IN HART, D; RICE, A M; VUKOVIC, S
PA (ORDE-N) ORDER OF SISTERS OF MERCY IN QUEENSLAND; (ORDE-N) CORP ORDER SISTERS OF MERCY IN QUEENSLAN
CYC 105
PI WO 2004020613 A1 20040311 (200422)* EN 43
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW
AU 2003254412 A1 20040319 (200462)
ADT WO 2004020613 A1 WO 2003-AU1113 20030829; AU 2003254412 A1 AU 2003-254412 20030829
FDT AU 2003254412 A1 Based on WO 2004020613
PRAI AU 2002-951082 20020830
AB WO2004020613 A UPAB: 20040331
NOVELTY - Generating a population of dendritic cells comprising culturing or expanding CD34+ precursor cells in the presence of one or more cytokines for a time and under conditions sufficient to allow the CD34+ precursor cells to differentiate into a population of dendritic cells, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:

(1) a method for differentiating a population of CD34+ precursor cells into dendritic cells;

(2) a method of inducing a gradient of differentiated dendritic cells from a population of CD34+ precursor cells;

(3) a method for proliferating a population of CD34+ precursor cells and differentiating the expanded population into dendritic cells;

(4) a method for differentiating a population of CD34+ precursor cells into a population of dendritic cells and proliferating the dendritic cells into an expanded population of dendritic cells; and

(5) methods for inducing a protective immune response against an autoimmune disease, cancer or a pathogen in a subject.

ACTIVITY - Antimicrobial; Immunosuppressive; Cytostatic.

No biological data given.

MECHANISM OF ACTION - Immunotherapy.

USE - The methods and dendritic cells are useful for inducing a protective immune response in a subject against pathogenic infections, autoimmune diseases and cancer (claimed).

Dwg.0/6

L37 ANSWER 5 OF 42 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN DUPLICATE 3
AN 2004-082868 [08] WPIDS
DNC C2004-034084
TI Modulating an immune response, useful for treating immune disorders, e.g. viral, bacterial and parasitic infections, prion diseases, or neoplastic diseases, administering to a subject an overlapping synthetic peptide formulation.
DC B04 C06 D16
IN JIANG, S; RUPRECHT, R M
PA (DAND) DANA FARBER CANCER INST INC
CYC 105
PI WO 2004002415 A2 20040108 (200408)* EN 175
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW

W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK
DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR
KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PG PH
PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC
VN YU ZA ZM ZW
AU 2003245729 A1 20040119 (200447)
ADT WO 2004002415 A2 WO 2003-US20322 20030627; AU 2003245729 A1 AU 2003-245729 20030627
FDT AU 2003245729 A1 Based on WO 2004002415
PRAI US 2002-392718P 20020627
AB WO2004002415 A UPAB: 20040202
NOVELTY - Modulating an immune response comprising administering to a subject an overlapping synthetic peptide formulation (OSPF).
DETAILED DESCRIPTION - Modulating an immune response comprising administering to a subject an overlapping synthetic peptide formulation (OSPF). The OSPF comprises a combination of single chain peptides corresponding to an amino acid sequence of a protein of interest, where the single chain peptide is a length represented by Y, which is at least 7 to (X-1), and X is the number of amino acids of the protein of interest. The single chain peptide overlaps with another single chain peptide by a length represented by Z, where Z is 1 to (Y-1), and the length of the single chain peptide is such that internalization of the single chain peptide by a major histocompatibility complex (MHC)-bearing cell and presentation by a MHC molecule to a T cell is possible.
INDEPENDENT CLAIMS are also included for the following:
(1) a pharmaceutical composition comprising OSPF defined above, and a pharmaceutical;
(2) treating or preventing an OSPF-associated disorder in a subject by administering an OSPF;
(3) a vaccine for immunizing a subject against an OSPF-associated disorder by modulating a CTL-mediated immune response, comprising a carrier, and an OSPF;
(4) a kit for immunizing a subject against an OSPF-associated disorder comprising the vaccine, and instructions for use;
(5) an adjuvant for a vaccine comprising a pharmaceutical carrier and an OSPF; and
(6) modulating an immune response comprising contacting a cell with an OSPF.
ACTIVITY - Immunostimulant; Virucide; Antibacterial; Antiparasitic; Cytostatic.
No biological data is given.
MECHANISM OF ACTION - Vaccine.
USE - The method is useful for treating immune disorders, e.g. viral, bacterial, and parasitic infections, prion diseases, neoplastic diseases, and protection against toxins.
Dwg.0/3

L37 ANSWER 6 OF 42 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
AN 2004-784471 [77] WPIDS
DNN N2004-618320 DNC C2004-274512
TI Diagnosing breast tumor, by detecting expression product of one of 119 genes encoding, for example, ribosomal protein L27 and HIF-1 responsive RTP801, in breast tissue where increased expression indicates neoplastic state.
DC B04 D16 P31 S03
IN MADDEN, S; SUKUMAR, S
PA (MADD-I) MADDEN S; (SUKU-I) SUKUMAR S
CYC 108
PI WO 2004091383 A2 20041028 (200477)* EN 50
RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE

LS LU MC MW MZ NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE
DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG
KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NA NI NO NZ
OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG
US UZ VC VN YU ZA ZM ZW

ADT WO 2004091383 A2 WO 2004-US9704 20040331
PRAI US 2003-458960P 20030401
AB WO2004091383 A UPAB: 20041203

NOVELTY - Method (M1) to aid in diagnosing breast tumor, by detecting expression product of any one of 119 gene (such as hypothetical protein DKFZp434G171, HIF-1 responsive RTP801, ribosomal protein L27, cyclin-dependent kinase 3) in first breast tissue sample suspected of neoplastic, and comparing expression of gene in second breast tissue sample which is normal, where increased expression of gene in first sample indicates neoplastic state.

DETAILED DESCRIPTION - Method (M1) to aid in diagnosing breast tumor, involves detecting an expression product of at least any one of 119 gene in first breast tissue sample suspected of neoplastic, where the gene includes hypothetical protein DKFZp434G171, heat shock 70 kDa protein 1A, jagged 1 (Alagille syndrome), cyclin-dependent kinase 3, 6-phosphogluconolactonase, homolog of rat and mouse retinoid-inducible serine carboxypeptidase, plasmalemma vesicle associated protein, NADH:ubiquinone oxidoreductase MLRQ subunit homolog, HIF-1 responsive RTP801, ribosomal protein L27, etc. and comparing the expression of at least one gene in the first breast tissue sample with expression of at least one gene in the second breast tissue sample which is normal, where increased expression of at least one gene in the first breast tissue sample relative to the second tissue sample identifies the first breast tissue sample to be neoplastic.

INDEPENDENT CLAIMS are also included for the following:

(1) treating (M2) a breast tumor, involves contacting the cells of the breast tumor with an antibody that specifically binds to an extracellular epitope of a protein selected from benzodiazapine receptor (peripheral); cadherin 5, type 2, VE-cadherin (vascular epithelium), calcium channel, voltage-dependent, alpha 1H subunit; CD74 antigen (invariant polypeptide of major histocompatibility complex, class 1:1 antigen associated); CD9 antigen (p24); dysferlin, limb girdle muscular dystrophy 2B (autosomal recessive), ectonucleoside triphosphate diphosphohydrolase 1, G protein-coupled receptor 4, hypothetical protein FLJ20898, hypoxia up-regulated 1, immediate early response 3, interferon, alpha-inducible protein (clone IFI-6-16), jagged 1 (Alagille syndrome), KLA,A0152 gene product, Lysosomal-associated multispanning membrane protein-5, major histocompatibility complex, class I, B, major histocompatibility complex, class I, C, NADH:ubiquinone oxidoreductase MLRQ subunit homolog, Notch homolog 3 (Drosophila), plasmalemma vesicle associated protein, solute carrier family 21 (prostaglandin transporter), member 2, TEMB, Thy-I cell surface antigen, receptor (calcitonin) activity modifying protein 3, sema domain, immunoglobulin domain (Ig), 43 benzodiazapine receptor (peripheral) - mitochondrial, and TEM17, where immune destruction of cells of the breast tumor is triggered;

(2) identifying (M3) the test compound as potential anti-cancer or anti-breast tumor drug, involves contacting a test compound with a cell expressing at least one gene of (M1), monitoring an expressing product of the gene, and identifying the test compound as a potential anti-cancer drug if it decreases the expression of at least one gene; and

(3) inducing (M4) an immune response to a breast tumor, involves administering to a mammal a protein or nucleic acid encoding a protein of (M1), where an immune response to the protein is induced.

ACTIVITY - Cytostatic; Immunostimulant.

No supporting data is given.

MECHANISM OF ACTION - Immunotoxin; Radioimmunotherapeutic.

USE - (M1) is useful for diagnosing breast tumor. The tissue samples are isolated from same human. (M2) is useful for treating breast tumor. (M4) is useful for inducing an immune response to a breast tumor in a mammal. The mammal has a breast tumor. The mammal has a breast tumor that is surgically removed (all claimed).

ADVANTAGE - (M1) provides distinct diagnosis of neoplastic and normal endothelium in human breast at molecular level and has significant implication for the development of anti-angiogenic therapies.

Dwg.0/0

L37 ANSWER 7 OF 42 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
AN 2004-203791 [19] WPIDS
DNC C2004-080454
TI Controlling secretions from holocrine glands, or holocrine-like components of cerumen and **mammary** glands, comprises administration of **botulinum** toxin.
DC B04
IN AQUILA, R; SANDERS, I
PA (SAND-I) SANDERS I
CYC 105
PI WO 2004016763 A2 20040226 (200419)* EN 27
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW
AU 2003263860 A1 20040303 (200457)
ADT WO 2004016763 A2 WO 2003-US25708 20030818; AU 2003263860 A1 AU 2003-263860 20030818
FDT AU 2003263860 A1 Based on WO 2004016763
PRAI US 2002-404378P 20020819
AB WO2004016763 A UPAB: 20040318

NOVELTY - Controlling secretions from holocrine glands, or holocrine-like components of cerumen and **mammary** glands in patients whose level of glandular secretion is greater than is desirable comprises administering to the patient a secretorily controlling amount of **botulinum** toxin.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is included for a method for smoothing fine wrinkles in the skin and decreasing the skin pore size of a subject in need of the same, comprising administering to the patient the botulinum toxin.

ACTIVITY - Antiinflammatory; Antibacterial; Dermatological; Antiseborrheic.

Test details are described but no results are given.

MECHANISM OF ACTION - None given.

USE - The method is useful for controlling secretions in patient having a condition resulting from greater than the desirable level of secretion selected from seborrheic dermatitis, rhinophyma, seborrheic blepharitis, sebaceous cysts, excess cerumen, unwanted milk production, and bacterial infections of these glands resulting in hidradenitis, furuncles, carbuncles, styes, and chalazions. The botulinum toxin is useful for smoothing wrinkles in the skin and decreasing the skin pore size of the subject (all claimed).

Dwg.0/0

L37 ANSWER 8 OF 42 HCAPLUS COPYRIGHT 2005 ACS on STN

AN 2004:515671 HCAPLUS
DN 141:66293
TI Protein and cDNA sequences of a novel human cancer gene BASE, and therapeutic use
IN Pastan, Ira H.; Egland, Kristi A.; Vincent, James J.; Lee, Byungkook; Strausberg, Robert
PA United States Dept. of Health and Human Services, USA
SO PCT Int. Appl., 86 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004053098	A2	20040624	WO 2003-US39476	20031210

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 2002-432531P P 20021210

AB The invention relates to the discovery of a new gene, termed 'BASE,' which is expressed in some 25% of breast cancers and in salivary glands. BASE is expressed in two alternatively spliced forms: a 19.5 kD, 179 amino acid secreted protein called 'base1,' and a 8.4 CKD, 79 amino acid non-secreted protein called 'base2.' The invention provides antibodies to base 1 and to base2. Antibodies to the proteins can be used to detect the presence of base 1 or base2 in a sample, thereby detecting the presence of a BASE-expressing breast cancer. Antibodies to base2 attached to a therapeutic agent can direct the agent to base2-expressing cells. Base1 and base2, immunogenic fragments of the proteins, and analogs of the proteins can be used to raise immune responses to BASE-expressing cancer cells. The invention further provides uses for using the proteins in manufacturing medicaments and methods for using antibodies to the proteins, attached to therapeutic mols., to inhibit the growth of cancer cells expressing BASE.

L37 ANSWER 9 OF 42 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2005:60754 HCAPLUS
Correction of: 2004:1036571
DN 142:233342
Correction of: 142:16836
TI Sequences of human schizophrenia related genes and use for diagnosis, prognosis and therapy
IN Liew, Choong-Chin
PA Chondrogene Limited, Can.
SO U.S. Pat. Appl. Publ., 156 pp., Cont.-in-part of U.S. Ser. No. 802,875.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 39

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2004241727	A1	20041202	US 2004-812731	20040330
US 2004014059	A1	20040122	US 2002-268730	20021009

US 2004248169 A1 20041209 US 2004-812737 20040330
US 2004265869 A1 20041230 US 2004-812716 20040330
WO 2004112589 A2 20041229 WO 2004-US20836 20040621
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 1999-115125P P 19990106
US 2000-477148 B1 20000104
US 2002-268730 A2 20021009
US 2003-601518 A2 20030620
US 2004-802875 A2 20040312
US 2001-271955P P 20010228
US 2001-275017P P 20010312
US 2001-305340P P 20010713
US 2002-85783 A2 20020228
US 2004-809675 A 20040325

AB The present invention is directed to detection and measurement of gene transcripts and their equivalent nucleic acid products in blood. Specifically provided is anal. performed on a drop of blood for detecting, diagnosing and monitoring diseases using gene-specific and/or tissue-specific primers. The present invention also describes methods by which delineation of the sequence and/or quantitation of the expression levels of disease-specific genes allows for an immediate and accurate diagnostic/prognostic test for disease or to assess the effect of a particular treatment regimen. [This abstract record is one of 3 records for this document necessitated by the large number of index entries required to fully index the document and publication system constraints.].

L37 ANSWER 10 OF 42 EMBASE
on STN
AN 2004486402 EMBASE
TI Benefit-risk assessment of tolterodine in the treatment of overactive bladder in adults.
AU Garely A.D.; Burrows L.
CS Dr. A.D. Garely, 120 Mineola Boulevard, Mineola, NY 11501, United States. agarely@winthrop.org
SO Drug Safety, (2004) 27/13 (1043-1057).
Refs: 65
ISSN: 0114-5916 CODEN: DRSAEA
CY New Zealand
DT Journal; General Review
FS 010 Obstetrics and Gynecology
028 Urology and Nephrology
037 Drug Literature Index
038 Adverse Reactions Titles
039 Pharmacy
LA English
SL English
AB Overactive bladder is associated with symptoms of urgency, with or without urge incontinence, usually with daytime frequency and nocturia in the absence of local pathological factors. Muscarinic receptor antagonists (antimuscarinics) are the first-line pharmacotherapy. Tolterodine, a

competitive, nonselective antimuscarinic specifically developed for the treatment of overactive bladder, demonstrated tissue selectivity for the bladder over the parotid gland in an animal model. As of March 5, 2003, the immediate-release (IR) formulation had been approved in 72 countries and the extended-release (ER) formulation had been approved in 28 countries, and tolterodine had been administered to 5 million patients. This review evaluates the benefit-risk profile of tolterodine in the treatment of adults with overactive bladder, summarising clinical trial and postmarketing surveillance data. Tolterodine has been found to significantly reduce micturition frequency, urgency perception and the number of episodes of urge incontinence and increase the volume voided per micturition. Dry mouth, an antimuscarinic class effect, is the most commonly reported adverse effect but is mostly mild to moderate in severity. Serious adverse effects are reported infrequently. Based on summary and review of postmarketing surveillance and clinical trial safety data received by the market authorisation holder and contained in the Periodic Safety Update Reports for tolterodine, several monitored serious events of the gastrointestinal tract (e.g. ileus or haemorrhage), nervous system (e.g. syncope, convulsions and memory disorders) and cardiovascular system (e.g. ventricular arrhythmia, atrial fibrillation, palpitations, bradycardia, transient ischaemic attacks and hypertension) were not considered related to tolterodine. QT or corrected QT (QTc) prolongation was not observed in any of the five cases of verified ventricular arrhythmia in patients administered tolterodine; there is insufficient evidence to indicate that tolterodine causes ventricular arrhythmia or extrasystoles or any specific type of cardiac rhythm abnormality. The safety profile of tolterodine is similar in patients aged ≥65 years and in younger adults. Clinically relevant drug interactions are limited to cytochrome P450 3A4 inhibitors, such as ketoconazole, and co-administration with such agents warrants a tolterodine dosage decrease. In addition, tolterodine IR 2mg twice daily is similar in efficacy to oxybutynin IR 5mg three times daily, and tolterodine ER 4mg once daily is similar in efficacy to oxybutynin ER 10mg once daily. Dry mouth occurred less frequently with tolterodine than oxybutynin, and moderate to severe dry mouth occurred more than three times less frequently. Based on the low frequency of adverse events, the absence of unexpected adverse events and the very low frequency of serious adverse events, we conclude that tolterodine is a well tolerated treatment for overactive bladder in adults, in whom it should be considered as first-line therapy.

L37 ANSWER 11 OF 42 EMBASE
on STN
AN 2004409133 EMBASE
TI A 48-year-old woman with nausea, vomiting, early satiety, and weight loss.
AU Qadeer M.A.; Burke C.A.
CS Dr. C.A. Burke, Dept. of Gastroenterology/Hepatology, The Cleveland Clinic Foundation, 9500 Euclid Avenue, Cleveland, OH 44195, United States. burkec1@ccf.org
SO Cleveland Clinic Journal of Medicine, (2004) 71/9 (693-712).
Refs: 34
ISSN: 0891-1150 CODEN: CCJMEL
CY United States
DT Journal; General Review
FS 030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
048 Gastroenterology
LA English

L37 ANSWER 12 OF 42 MEDLINE on STN

AN 2004491426 MEDLINE
DN PubMed ID: 15383788
TI Botulinum toxin infiltration for pain control after mastectomy and expander reconstruction.
AU Layeeque Rakhshanda; Hochberg Julio; Siegel Eric; Kunkel Kelly; Kepple Julie; Henry-Tillman Ronda S; Dunlap Melinda; Seibert John; Klimberg V Suzanne
CS Department of Surgery, Division of Breast Surgical Oncology, University of Arkansas for Medical Sciences, Arkansas Cancer Research Center, and the Central Arkansas Veterans Hospital System, Little Rock, Arkansas, USA.
SO Annals of surgery, (2004 Oct) 240 (4) 608-13; discussion 613-4. Journal code: 0372354. ISSN: 0003-4932.
CY United States
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 200410
ED Entered STN: 20041005
Last Updated on STN: 20041022
Entered Medline: 20041021
AB INTRODUCTION: We hypothesized botulinum toxin (BT) infiltration of the chest wall musculature after mastectomy would create a prolonged inhibition of muscle spasm and postoperative pain, facilitating tissue expander reconstruction. METHODS: An Institutional Review Board (IRB)-approved prospective study was conducted of all patients undergoing mastectomy with tissue expander placement during a 2-year period. Study patients versus controls had 100 units of diluted BT injected into the pectoralis major, serratus anterior, and rectus abdominis insertion. Pain was scored using a visual analog scale of 0 to 10. Wilcoxon rank sum test was used for continuous variables and the chi2 test for nominal level data to test for significance. RESULTS: Forty-eight patients were entered into the study; 22 (46%) with and 26 (54%) without BT infiltration. Groups were comparable in terms of age (55 +/- 11 years versus 52 +/- 10 years; P = 0.46), bilateral procedure (59% versus 61%; P = 0.86), tumor size (2 +/- 2 cm versus 2 +/- 3 cm; P = 0.4), expander size and volume (429 +/- 119 mL versus 510 +/- 138 mL; P = 0.5). The BT group did significantly better with pain postoperatively (score of 3 +/- 1 versus 7 +/- 2; P < 0.0001), during initial (score of 2 +/- 2 versus 6 +/- 3; P = 1.6 x 10(-6)), and final expansion (1 +/- 1 versus 3 +/- 2; P = 0.009). Volume of expansion per session was greater thus expansion sessions required less in the BT group (5 +/- 1 versus 7 +/- 3; P = 0.025). There was a significant increase in narcotic use in control patients in the first 24 hours (17 +/- 10 mg versus 3 +/- 3 mg; P < 0.0001), initial as well as final expansion periods (P = 0.0123 and 0.0367, respectively). One expander in the BT group versus 5 in the control group required removal (P = 0.13). There were no BT-related complications. CONCLUSION: Muscular infiltration of botulinum toxin for mastectomy and tissue expander placement significantly reduced postoperative pain and discomfort without complications.

L37 ANSWER 13 OF 42 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 2004401950 EMBASE
TI Treatment of bruxism in Huntington's disease with botulinum toxin [2].
AU Nash M.C.; Ferrell R.B.; Lombardo M.A.; Williams R.B.
CS Dr. M.C. Nash, Colby Center for Psychiatry, Adirondack Medical Center, Saranac Lake, NY, United States
SO Journal of Neuropsychiatry and Clinical Neurosciences, (2004) 16/3 (381-382).
Refs: 5

ISSN: 0895-0172 CODEN: JNCNE7
CY United States
DT Journal; Letter
FS 008 Neurology and Neurosurgery
037 Drug Literature Index
LA English

L37 ANSWER 14 OF 42 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
AN 2005:23549 BIOSIS
DN PREV200500020549
TI 2004 Annual Meeting and Congress of the Schweizerische Gesellschaft fuer Gynaekologie und Geburtshilfe (SGGG), Interlaken, Switzerland, June 24-26, 2004.
AU Anonymous
SO Gynaekologisch-Geburtshilfliche Rundschau, (June 2004) Vol. 44, No. 3, pp. 164-218. print.
Meeting Info.: 2004 Annual Meeting and Congress of the Schweizerische Gesellschaft fuer Gynaekologie und Geburtshilfe. Interlaken, Switzerland. June 24-26, 2004. Schweizerische Gesellschaft fuer Gynaekologie und Geburtshilfe.
ISSN: 1018-8843.
DT Conference; (Meeting)
Conference; (Meeting Summary)
LA German
ED Entered STN: 29 Dec 2004
Last Updated on STN: 29 Dec 2004
AB This meeting contains approximately 162 abstracts written in French, German and English, on gynecology and obstetrics. **Diseases** discussed include but are not limited to motor compulsive incontinence, vulvar Paget **disease**, ovarian **carcinoma**, **breast cancer**, chlamydia trachomatis, and uterine **cancer.** Treatment strategies, prevention and control, prevalence, drugs, pathology, and outcomes of these diseases were all discussed.

L37 ANSWER 15 OF 42 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN DUPLICATE 4
AN 2003-636741 [60] WPIDS
DNC C2003-174130
TI New antibody that specifically binds an antigenic epitope of an MRP9 polypeptide, useful for preparing a composition for treating breast cancer.
DC B04 D16
IN BERA, T K; LEE, B; PASTAN, I H
PA (USSH) US DEPT HEALTH & HUMAN SERVICES
CYC 102
PI WO 2003062446 A2 20030731 (200360)* EN 84
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT SD SE SI SK SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW
AU 2003209265 A1 20030902 (200422)
ADT WO 2003062446 A2 WO 2003-US1340 20030115; AU 2003209265 A1 AU 2003-209265 20030115
FDT AU 2003209265 A1 Based on WO 2003062446
PRAI US 2002-375121P 20020422; US 2002-350053P 20020117
AB WO2003062446 A UPAB: 20030919
NOVELTY - A new antibody which specifically binds an antigenic epitope of

an MRP9 polypeptide, comprising:
(a) a sequence comprising 931 amino acids, or its conservative variant;
(b) an immunogenic fragment comprising 8 consecutive amino acid residues of (a) that specifically binds to an antibody that specifically binds to (a); or
(c) a sequence that is at least 80% homologous to (a), having MRP9 activity, is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:
(1) a composition comprising the antibody and a carrier;
(2) kits for detecting an MRP9 polypeptide or for detecting a nucleic acid encoding the MRP9 polypeptide;
(3) detecting a cancer in a subject;
(4) producing an immune response against a neoplastic cell expressing MRP9 in a subject;
(5) inhibiting the growth of a neoplastic cell expressing MRP9; and
(6) a purified polypeptide having a sequence comprising 931 amino acids.
ACTIVITY - Cytostatic. No biological data given.
MECHANISM OF ACTION - Gene therapy.
USE - The antibody is useful for preparing a composition for treating breast cancer (claimed).
Dwg.0/7

L37 ANSWER 16 OF 42 MEDLINE on STN
AN 2003024662 MEDLINE
DN PubMed ID: 12531431
TI Molecular mechanism of the anti-cancer activity of cerivastatin, an inhibitor of HMG-CoA reductase, on aggressive human breast cancer cells.
AU Denoyelle Christophe; Albanese Patricia; Uzan Georges; Hong Li; Vannier Jean-Pierre; Soria Jeannette; Soria Claudine
CS Laboratoire DIFEMA, Groupe de Recherche MERCI, UFR de Medecine et de Pharmacie, 76183 Rouen, France.
SO Cellular signalling, (2003 Mar) 15 (3) 327-38.
Journal code: 8904683. ISSN: 0898-6568.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200308
ED Entered STN: 20030118
Last Updated on STN: 20030829
Entered Medline: 20030828
AB Statins are currently used for the treatment of hypercholesterolemia. Recently, we demonstrated that cerivastatin also reduces the proliferation and invasion of aggressive breast cancer cells, MDA-MB-231. In this report, a molecular mechanism to explain its anti-cancer action is proposed by combining the study of cerivastatin effect on both gene expression (microarray) and signal transduction pathways. Firstly, the expression of 13 genes was modified by cerivastatin and confirmed at protein level. They could contribute to the inhibition of both cell proliferation (down-regulation of cyclin D1, PCNA, c-myc and up-regulation p21(Waf1), p19(INK4d), integrin beta8) and cell invasion, either directly (decrease in u-PA, MMP-9, u-PAR, PAI-1 and increase in anti-oncogenes Wnt-5a and H-cadherin) or indirectly by stimulating an anti-angiogenic gene (thrombospondin-2). The anti-angiogenic activity was confirmed by in vivo experiments. Secondly, we demonstrated that the biochemical mechanism of its anti-cancer action could be mainly explained by the inhibition of RhoA-dependent cell signalling. This hypothesis was

supported by the fact that a RhoA inhibitor (C3 exoenzyme) or a dominant negative mutant RhoA (N19RhoA) induced similar effects to those of cerivastatin. In conclusion, cerivastatin, by preventing RhoA prenylation, inhibits (i) the RhoA/ROCK pathway, leading to defective actin stress fibres formation responsible for the loss of traction forces required for cell motility and (ii) the RhoA/FAK/AKT signalling pathway that could explain the majority of cancer-related gene modifications described above. Thus, the inhibition of RhoA cell signalling could be a good strategy in therapy of aggressive forms of breast cancer. Copyright 2002 Elsevier Science Inc.

L37 ANSWER 17 OF 42 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN DUPLICATE 5
AN 2003-018772 [01] WPIDS
CR 2002-463338 [49]
DNC C2003-004552
TI New agent comprising a light chain and a (modified) heavy chain of a **botulinum**, butyricum, or tetani toxin, useful for treating a gonadotrophin related illness, e.g. **breast**, prostate pancreatic or endometrial cancer, or endometriosis.
DC B04 D16
IN DONOVAN, S
PA (ALLR) ALLERGAN INC; (ALLR) ALLERGAN SALES INC
CYC 101
PI WO 2002074327 A2 20020926 (200301)* EN 97
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG US UZ VN YU ZA ZM ZW
US 2002177545 A1 20021128 (200302)
EP 1368053 A2 20031210 (200382) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR
AU 2002252284 A1 20021003 (200432)
JP 2004525922 W 20040826 (200456) 131
US 6831059 B2 20041214 (200501)
ADT WO 2002074327 A2 WO 2002-US7379 20020311; US 2002177545 A1 CIP of US 2000-692811 20001020, US 2001-810601 20010315; EP 1368053 A2 EP 2002-721347 20020311, WO 2002-US7379 20020311; AU 2002252284 A1 AU 2002-252284 20020311; JP 2004525922 W JP 2002-573034 20020311, WO 2002-US7379 20020311; US 6831059 B2 CIP of US 2000-692811 20001020, US 2001-810601 20010315
FDT EP 1368053 A2 Based on WO 2002074327; AU 2002252284 A1 Based on WO 2002074327; JP 2004525922 W Based on WO 2002074327
PRAI US 2001-810601 20010315; US 2000-692811 20001020
AB WO 200274327 A UPAB: 20050103
NOVELTY - A new agent comprising:

(a) a light chain component comprising a light chain or fragment of a botulinum, butyricum, or tetani toxin or their variants;

(b) a translocation component comprising a heavy chain or a modified heavy chain of a botulinum, butyricum, or tetani toxin or their variants; and

(c) a targeting component which selectively binds to a GnRH receptor, is new

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for a method for treating a gonadotrophin related illness in a mammal by administering to the mammal an agent described above.

ACTIVITY - Cytostatic; Gynecological.

A 54-year old male tests positive for prostate specific antigen (PSA). Patient was administered with LH N-GnRH directly to the anterior pituitary at a dose sufficient to reduce the patient's level of circulating gonadotrophin by 80% to 30%, preferably 50%. Patient was monitored closely for advance of the cancer. Over the next 24 months, there is no spread of the cancer and no detectable further enlargement of the prostate. Treatment was repeated at 27 months. At 36 months from the initial diagnosis, patient no longer tested positive for PSA.

MECHANISM OF ACTION - Gonadotrophin inhibitor.

USE - The agent is useful for treating a gonadotrophin related illness in a mammal, including a human, where gonadotrophin related illness is breast cancer, prostate cancer, pancreatic cancer, endometriosis, endometrial cancer, or precocious puberty (claimed). The agent is also useful for decreasing gonadotrophin secretion in a mammal. Dwg.0/1

L37 ANSWER 18 OF 42 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN DUPLICATE 6
AN 2002-362353 [39] WPIDS
DNC C2002-102590
TI New monoclonal antibody which specifically binds and forms complex with TIP-2 antigen located on surface of human cancer cells, useful for diagnosing and treating cancer in a human subject.
DC B04 D16
IN CANFIELD, R; KALANTAROV, G; RUDCHENKO, S; TRAKHT, I
PA (UYCO) UNIV COLUMBIA NEW YORK
CYC 97
PI WO 2002022851 A2 20020321 (200239)* EN 276
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PH PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW
AU 2001092782 A 20020326 (200251)
EP 1326894 A2 20030716 (200347) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR
JP 2004518630 W 20040624 (200442) 406
ADT WO 2002022851 A2 WO 2001-US29242 20010918; AU 2001092782 A AU 2001-92782 20010918; EP 1326894 A2 EP 2001-973176 20010918, WO 2001-US29242 20010918; JP 2004518630 W WO 2001-US29242 20010918, JP 2002-527293 20010918
FDT AU 2001092782 A Based on WO 2002022851; EP 1326894 A2 Based on WO 2002022851; JP 2004518630 W Based on WO 2002022851
PRAI US 2000-664958 20000918
AB WO 200222851 A UPAB: 20020621
NOVELTY - A monoclonal antibody (I) which specifically binds and forms a complex with TIP-2 antigen located on the surface of human cancer cells, where (I) binds to the same antigen as monoclonal antibody 27.B1 or 27 produced by hybridoma 27.B1 or 27 of ATCC Designation Number PTA-1599 or 1598, respectively, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a hybridoma cell (II) producing (I);

(2) treating (M1) cancer in a human subject involves:

(a) evoking a specific immune response by administering to the subject a whole TIP-2 antigen protein or its peptide fragment to the subject, or by removing dendritic cells from the subject, contacting the dendritic cells with a whole TIP-2 antigen protein or its peptide and reintroducing the dendritic cells into the subject; or

(b) inducing apoptosis of cancer cells, by administering to the

subject a whole TIP-2 antigen protein or its peptide fragment to the subject;

(3) an isolated peptide (III) having the sequence Lys-Leu-Leu-Gly-Gly-Gln-Ile-Gly-Leu or Ser-Leu-Leu-Gly-Cys-Arg-His-Tyr-Glu-Val;

(4) a kit (IV) for detecting the presence of TIP-2 antigen-bearing cancer cells in a sample, comprises a solid support having several covalently linked probes which may be the same or different, each probe of which comprises a monoclonal antibody directed to an epitope on TIP-2 antigen or its Fab fragment, and unit for determining the presence of monoclonal antibody/Fab fragment-TIP-2 antigen complex;

(5) diagnosing (M2) cancer associated with the expression of TIP-2 antigen in a human subject, involves:

(a) obtaining mRNA from a sample of the subject's peripheral blood, preparing cDNA from the mRNA, amplifying DNA encoding TIP-2 antigen present in the cDNA by a polymerase chain reaction (PCR) utilizing at least two oligonucleotide primers, where each of the primer specifically hybridizes with DNA encoding TIP-2 antigen, where the primers comprise oligonucleotides having a sequence as given in the specification, and detecting the presence of any resulting amplified DNA, where the presence of such amplified DNA is diagnostic for cancer associated with the expression of TIP-2 antigen; or

(b) obtaining mRNA from a sample of the subject's peripheral blood, preparing cDNA from the mRNA, amplifying DNA encoding TIP-2 antigen present in the cDNA, determining the amount of any resulting amplified DNA, and comparing the amount of amplified DNA determined with previously determined standard amounts of amplified DNA, where each standard amount is indicative of a particular stage of cancer associated with the expression of TIP-2 antigen; and

(6) a composition (V) which comprises a suitable carrier and a monoclonal antibody produced by fusing a lymphoid cell capable of producing antibody with a trioma cell which does not produce any antibody and is obtained by fusing a heteromyeloma cell which does not produce any antibody with a human lymphoid cell so as to form tetroma cells, incubating the tetroma cells under conditions permissive for the production of antibody by the tetroma cells, to produce the monoclonal antibody and recovering the monoclonal antibody so produced.

ACTIVITY - Cytostatic; antitumor; dermatological; antithyroid; immunosuppressive; antirheumatic; antiarthritic; antibacterial; virucide.

MECHANISM OF ACTION - Inducer of apoptosis of TIP-2 antigen bearing cells (claimed). No supporting data is given.

USE - (I) is useful for detecting TIP-2 antigen bearing cancer cells, for diagnosing cancer in a subject by detecting TIP-2 antigen-bearing cancer cells, for in vivo diagnosis of cancer in a subject, for delivering exogenous material to TIP-2 antigen-bearing cancer cells of a human subject, for treating cancer in a human subject, for inducing apoptosis of TIP-2 antigen bearing cells, for immunohistochemical screening of a tissue section from a tumor sample for the presence of TIP-2 antigen bearing cancer cells, for detecting the presence of TIP-2 antigen in biological fluid, and for monitoring progression of cancer, where the cancer cells are TIP-2 antigen-bearing cancer cells, in a subject. (V) is useful for treating or preventing a condition in a subject who previously exhibited the condition, where the condition is associated with cancer (thyroid, **breast** or prostate cancer), tumor (benign), toxin (tetanus, anthrax, **botulinum**, snake venom or spider venom), infectious agent (such as Hanta virus, HTLV I, HTLV II, HIV, herpes virus, influenza, Ebola, human papilloma virus, Staphylococcus, Streptococcus, Klebsiella, Escherichia coli, anthrax or Cryptococcus), enzyme dysfunction (hyperactivity or overproduction of the enzyme), hormone dysfunction (hyperactivity or overproduction of the hormone), autoimmune disease

(lupus, thyroiditis, graft versus host disease, transplantation rejection or rheumatoid arthritis), immune dysfunction (CD3 or CD4 mediated), viral antigen, bacterial antigen, eukaryotic antigen, rejection of a transplanted tissue, or the condition is septicemia, sepsis, septic shock, viremia, bacteremia, fungemia (claimed).
Dwg.0/42

L37 ANSWER 19 OF 42 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN DUPLICATE 7
AN 2002-673634 [72] WPIDS
CR 2001-006327 [01]; 2002-179993 [23]; 2002-254424 [30]; 2002-453014 [48]; 2005-131969 [14]
DNC C2002-189747
TI Treatment of a mammary gland disorder involves use of clostridial neurotoxin.
DC B04
IN BRIN, M F; DONOVAN, S
PA (ALLR) ALLERGAN INC; (ALLR) ALLERGAN SALES INC
CYC 103
PI US 2002094339 A1 20020718 (200272)* 19
WO 2004071525 A1 20040826 (200456) EN
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT SD SE SI SK SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG UZ VC VN YU ZA ZM ZW
AU 2003225549 A1 20040906 (200480)
EP 1492561 A1 20050105 (200504) EN
R: AL AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IT LI LT LU LV MC MK NL PT RO SE SI SK TR
ADT US 2002094339 A1 CIP of US 1999-454842 19991207, CIP of US 2000-631221 20000802, US 2002-71826 20020208; WO 2004071525 A1 WO 2003-US3479 20030204; AU 2003225549 A1 AU 2003-225549 20030204; EP 1492561 A1 EP 2003-815338 20030204, WO 2003-US3479 20030204
FDT US 2002094339 A1 CIP of US 6139845; AU 2003225549 A1 Based on WO 2004071525; EP 1492561 A1 Based on WO 2004071525
PRAI US 2002-71826 20020208; US 1999-454842 19991207;
US 2000-631221 20000802
AB US2002094339 A UPAB: 20050228
NOVELTY - Treating a **mammary** gland disorder involves local administration of clostridial neurotoxin (preferably **botulinum** toxin) (10-3 - 2000 U/kg).

ACTIVITY - Cytostatic.

MECHANISM OF ACTION - None given.

USE - For treating a mammary gland disorder including precancerous breast tissue, cystic breast cancer, carcinoma, breast cyst, sclerosing adenosis, duct papilloma, fibroadenoma, blunt duct adenosis and proliferative breast disease; preventing development of a mammary gland neoplasm (all claimed). Also useful for treating lung cancer, adencarcinomas, ovarian cancer, oral and oropharyngeal cancer, pancreatic cancer, prostate cancer, kidney cancer and testicular cancer.

ADVANTAGE - (A) provides an effective and long lasting therapeutic relief. (A) reduces size and/or activity of hyperplastic, hypertonic or neoplastic mammary gland tissue; reduces the diameter of the hyperplastic, hypertonic or neoplastic mammary gland tissue by about 20 - 100%; reduces secretion from the hyperplastic tissue comprising a substrate selected from vesicle membrane docking proteins consisting of synaptosomal associated protein (SNAP-25) (25 kiloDalton), synaptobrevin and syntaxin by inhibiting a vesicle mediated exocytosis from precancerous hyperplastic

tissue or hypertonic mammary tissue.
Dwg.0/0

L37 ANSWER 20 OF 42 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2002:905925 HCAPLUS
DN 138:8325
TI Vector for targeted delivery to cells
IN Medina-Kauwe, Lali K.; Kedes, Larry H.; Kasahara, Nori
PA University of Southern California, USA
SO PCT Int. Appl., 47 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002094318	A1	20021128	WO 2002-US16111	20020520

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 2001-292192P P 20010518
AB A non-viral single fusion protein vector for targeted cellular delivery which comprises a cell-targeting moiety, such as herugulin; a cell penetration penton moiety; and optionally a polynucleotide binding moiety, such as a polylysine sequence. The vector may further comprise an active agent, such as a therapeutic agent. Compns. comprising the vector and methods of utilizing the compns. are also provided.
RE.CNT 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L37 ANSWER 21 OF 42 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2002:172086 HCAPLUS
DN 136:214954
TI A cancer-associated gene XAGE-1 and its two encoded proteins, and therapeutic uses thereof in cancer treatment
IN Pastan, Ira H.; Liu, Xiu Fen; Bera, Tapan K.; Lee, Byungkook; Egland, Kristi A.
PA United States Dept. of Health and Human Services, USA
SO PCT Int. Appl., 79 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002018584	A2	20020307	WO 2001-US27258	20010831

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,

DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

	AU 2001087004	A5	20020313	AU 2001-87004	20010831
	US 2004087772	A1	20040506	US 2003-363233	20030304
PRAI	US 2000-229684P	P	20000901		
	WO 2001-US27258	W	20010831		

AB The invention relates to the surprising discovery that XAGE-1 is translated as two proteins, a 9 kDa protein, termed p9, and a 16.3 kDa protein, termed p16. XAGE-1 gene is cloned from Ewing's sarcoma and expressed sequence tag (EST) database anal. indicates that XAGE-1 is frequently found in Ewing's sarcoma and alveolar rhabdomyosarcoma. The invention further relates to the surprising discovery that XAGE-1 is expressed in a number of important human cancers, specifically: prostate cancer, lung cancer, ovarian cancer, breast cancer, glioblastoma, pancreatic cancer, T cell lymphoma, melanoma, and histocytic lymphoma. The proteins p9 and p16, immunogenic fragments thereof, analogs of these proteins, and nucleic acids encoding these proteins, fragments, or analogs, can be administered to persons with XAGE-1 expressing cancers to raise or augment an immune response to the cancer. The gene is located on the X chromosome. It encodes two proteins p16 and p9 (named after the mol. weight), and p9 is a shorter version of p16 only missing 66-amino acid at the N-terminal end. The encoded proteins share homol. with GAGE/PAGE proteins in their COOH-terminal ends. The invention further provides nucleic acid sequences encoding the proteins, as well as expression vectors, host cells, and antibodies to the proteins. Further, the invention provides immunoconjugates that comprise an antibody to p16 or to p9, and an effector mol., such as a label, a radioisotope, or a toxin. The invention also provides methods of inhibiting the growth of XAGE-1 expressing cells by contacting them with immunoconjugates comprising an anti-p9 or p16 antibody and a toxic moiety. Further, the invention provides kits for detecting the presence of p9 or p16 in a sample. These findings could be valuable for cancer diagnosis and cancer immunotherapy. The authors' previous expressed sequence tag database anal. indicates that XAGE-1 is frequently found in Ewing's sarcoma and alveolar rhabdomyosarcoma. Using Northern blots and RNA dot blots, the authors have now found that XAGE-1 is highly expressed in normal testis, in seven of eight Ewing's cell lines, in four of nine Ewing's sarcoma patient samples, and in one of one alveolar rhabdomyosarcoma patient sample. The gene is located on the X chromosome. The full-length cDNA contains 611 bp and predicts a protein of Mr 16,300 with a potential transmembrane domain at the NH2 terminus. XAGE-1 shares homol. with GAGE/PAGE proteins in the COOH-terminal end. These findings could be valuable for cancer diagnosis and cancer immunotherapy.

L37 ANSWER 22 OF 42 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2002:171732 HCAPLUS
DN 136:215419
TI Sensitization of cancer cells to immunotoxin-induced cell death by transfection with interleukin-13 receptor α2 chain
IN Puri, Raj K.
PA United States Dept. of Health and Human Services, USA
SO PCT Int. Appl., 80 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002017968	A2	20020307	WO 2001-US25663	20010815
	WO 2002017968	A3	20020418		

WO 2002017968 C2 20020704
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
AU 2001084978 A5 20020313 AU 2001-84978 20010815
US 2004136959 A1 20040715 US 2003-250998 20030708
PRAI US 2000-229842P P 20000831
WO 2001-US25663 W 20010815

AB The author discloses that cancer cells that have little or no expression of the IL-13 receptor (IL-13R) can bind IL-13R-targeted immunoconjugates, such as immunotoxins, after transfection with the IL-13R α2 chain. For some cancers, transfection with the IL-13R α2 chain alone inhibits tumor growth. In one example, using a plasmid vector, pancreatic cancer cells were transfected with IL-13R α2 chain. The transfected cells showed enhanced binding to the IL-13 ligand and became susceptible to the cytotoxic activity of an IL-13-Pseudomonas exotoxin chimera.

L37 ANSWER 23 OF 42 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2002:907158 HCAPLUS
DN 138:665
TI Compositions and methods for treating gonadotrophin related illnesses
IN Donovan, Stephen
PA Allergan Sales, Inc., USA; Allergan, Inc.
SO U.S. Pat. Appl. Publ., 36 pp., Cont.-in-part of U. S. Ser. No. 692,811.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2002177545	A1	20021128	US 2001-810601	20010315
	US 6831059	B2	20041214		
	US 6827931	B1	20041207	US 2000-692811	20001020
	ES 2218444	T3	20041116	ES 2001-1964282	20010821
	WO 2002074327	A2	20020926	WO 2002-US7379	20020311
	WO 2002074327	A3	20021212		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	EP 1368053	A2	20031210	EP 2002-721347	20020311
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	JP 2004525922	T2	20040826	JP 2002-573034	20020311
PRAI	US 2000-692811	A2	20001020		
	US 2001-810601	A	20010315		
	WO 2002-US7379	W	20020311		

OS MARPAT 138:665

AB The present invention relates to an agent comprising a neurotoxin, methods for making the agents and methods for treating endocrine disorders, for example gonadotrophin-related illnesses. Preferably, the agent comprises at least a portion of a botulinum toxin.

L37 ANSWER 24 OF 42 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN DUPLICATE 8
AN 2002:550646 BIOSIS
DN PREV200200550646
TI Rho GTPases in human breast tumours: Expression and mutation analyses and correlation with clinical parameters.
AU Fritz, G. [Reprint author]; Brachetti, C.; Bahlmann, F.; Schmidt, M.; Kaina, B.
CS Division of Applied Toxicology, Institute of Toxicology, University of Mainz, Obere Zahlbacher Str. 67, D-55131, Mainz, Germany fritz@mail.uni-mainz.de
SO British Journal of Cancer, (9 September, 2002) Vol. 87, No. 6, pp. 635-644. print.
CODEN: BJCAAI. ISSN: 0007-0920.
DT Article
LA English
ED Entered STN: 23 Oct 2002
Last Updated on STN: 23 Oct 2002
AB In the present study, we addressed the question of a putative relevance of Rho proteins in tumour progression by analysing their expression on protein and mRNA level in breast tumours. We show that the level of RhoA, RhoB, Rac1 and Cdc42 protein is largely enhanced in all tumour samples analysed (n=15) as compared to normal tissues originating from the same individual. The same is true for 32P-ADP-ribosylation of Rho proteins which is catalysed by Clostridium **botulinum** exoenzyme C3. Also the amount of Rho-GDI and ERK2 as well as the level of overall 32P-GTP binding acvitity was tumour-specific elevated, yet to a lower extent than Rho proteins. Although the amount of Rho proteins was enhanced in tumours, most of them did not show changes in rho mRNA expression as compared to the corresponding normal tissue. Thus, elevated gene expression seems not to be the underlying mechanism of tumour-specific overexpression of Rho proteins. Sequence analysis of RhoA, RhoB, RhoC and Rac1 failed to detect any mutations in both the GTP-binding site and effector binding region. By analysing>50 tumour samples, the amount of RhoA-like proteins (i.e. RhoA, B, C), but not of Rac1, was found to significantly increase with histological grade and proliferation index. Rho protein expression was neither related to p53 nor to HER-2/neu oncogene status. Expression of rho mRNAs did not show a significant increase with histological grade. Overall the data show that (1) Rho proteins are overexpressed in breast tumours (2) overexpression is not regulated on the mRNA level (3) the expression level of RhoA-like proteins correlates with malignancy and (4) Rho proteins are not altered by mutation in breast tumours.

L37 ANSWER 25 OF 42 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
AN 2002:394968 BIOSIS
DN PREV200200394968
TI CD44 function as receptor and effector on signaling by its ligand stimulation in Rho GTPase-meditated cell motility.
AU Higashi, Morihiro [Reprint author]; Kumagai, Shinpei [Reprint author]; Kitagawa, Motoo [Reprint author]; Sugimoto, Katsumi [Reprint author]; Kasagawa, Takahiro [Reprint author]; Harigaya, Kenichi [Reprint author]
CS Graduate School of Medicine, Molecular Tumor Pathology, Chiba University, Chiba, Japan

SO Proceedings of the American Association for Cancer Research Annual Meeting, (March, 2002) Vol. 43, pp. 371. print.
Meeting Info.: 93rd Annual Meeting of the American Association for Cancer Research. San Francisco, California, USA. April 06-10, 2002.
ISSN: 0197-016X.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 24 Jul 2002
Last Updated on STN: 24 Jul 2002

L37 ANSWER 26 OF 42 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 2002333064 EMBASE
TI Lessons from the Women's Health Initiative: Primary prevention and gender health.
AU Day A.
CS Dr. A. Day, S./Women's Coll. Hlth. Sci. Centre, Women's College Campus, 76 Grenville St., Toronto, Ont. M5S 1B2, Canada. a.day@utoronto.ca
SO Canadian Medical Association Journal, (2002) 167/4 (361-362).
Refs: 9
ISSN: 0820-3946 CODEN: CMAJAX
CY Canada
DT Journal; Note
FS 010 Obstetrics and Gynecology
017 Public Health, Social Medicine and Epidemiology
037 Drug Literature Index
038 Adverse Reactions Titles
LA English

L37 ANSWER 27 OF 42 MEDLINE on STN
AN 2002347530 MEDLINE
DN PubMed ID: 12090470
TI Mitogen activated protein kinase pathway is involved in RhoC GTPase induced motility, invasion and angiogenesis in inflammatory breast cancer.
AU van Golen Kenneth L; Bao Li Wei; Pan Quintin; Miller Fred R; Wu Zhi Fen; Merajver Sofia D
CS Department of Internal Medicine, University of Michigan Comprehensive Cancer Center, Ann Arbor 48109-0948, USA.
NC 5T32 CA 09537 (NCI)
R01 CA 77612 (NCI)
SO Clinical & experimental metastasis, (2002) 19 (4) 301-11.
Journal code: 8409970. ISSN: 0262-0898.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200207
ED Entered STN: 20020702
Last Updated on STN: 20021219
Entered Medline: 20020719
AB Inflammatory breast cancer (IBC) is the most lethal form of locally advanced breast cancer known. IBC carries a guarded prognosis primarily due to rapid onset of disease, typically within six months, and the propensity of tumor emboli to invade the dermal lymphatics and spread systemically. Although the clinical manifestations of IBC have been well documented, until recently little was known about the genetic mechanisms underlying the disease. In a comprehensive study aimed at identifying the molecular mechanisms responsible for the unique IBC phenotype, our laboratory identified overexpression of RhoC GTPase in over 90% of IBC

tumors in contrast to 36% of stage-matched non-IBC tumors. We also demonstrated that overexpression of RhoC GTPase in human mammary epithelial (HME) cells nearly recapitulated the IBC phenotype with regards to invasion, motility and angiogenesis. In the current study we sought to delineate which signaling pathways were responsible for each aspect of the IBC phenotype. Using well-established inhibitors to the mitogen activated protein kinase (MAPK) and phosphatidylinositol-3 kinase (PI3K) pathways. We found that activation of the MAPK pathway was responsible for motility, invasion and production of angiogenic factors. In contrast, growth under anchorage independent conditions was dependent on the PI3K pathway.

L37 ANSWER 28 OF 42 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 2002020668 EMBASE
TI Complex regional pain syndrome post mastectomy.
AU Graham L.E.; McGuigan C.; Kerr S.; Taggart A.J.
CS L.E. Graham, Registrar in Rheumatology, Musgrave Park Hospital, Stockman's Lane, Belfast BT9 7JB, United Kingdom. lorradam@wlink.com.np
SO Rheumatology International, (2002) 21/4 (165-166).
Refs: 13
ISSN: 0172-8172 CODEN: RHINDE
CY Germany
DT Journal; Article
FS 008 Neurology and Neurosurgery
030 Pharmacology
037 Drug Literature Index
LA English
SL English
AB Complex regional pain syndrome includes the previously termed condition reflex sympathetic dystrophy. It is a chronic pain disorder diagnosed on the basis of symptoms and skin changes and is known to have a psychological element. It is a rare complication after surgery, especially mastectomy. We present two females who developed this syndrome after undergoing mastectomy for chronic mastalgia. These cases demonstrate that amputation of an organ for chronic pain can result in reflex sympathetic dystrophy developing in a nearby limb.

L37 ANSWER 29 OF 42 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 2002298771 EMBASE
TI A variety of gamebird diseases reported in June.
SO Veterinary Record, (10 Aug 2002) 151/6 (161-164).
ISSN: 0042-4900 CODEN: VETRAX
CY United Kingdom
DT Journal; Note
FS 004 Microbiology
052 Toxicology
LA English

L37 ANSWER 30 OF 42 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN DUPLICATE 9
AN 2001-465198 [50] WPIDS
DNC C2001-140441
TI Treatment of pain associated with an interior disease site, involves administering a pain-relieving target construct to the patient.
DC B05 D16
IN LUIKEN, G A
PA (FLUO-N) FLUORO PROBE INC
CYC 94
PI WO 2001047512 A2 20010705 (200150)* EN 31
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ

NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM
DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC
LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE
SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2001049041 A 20010709 (200164)
ADT WO 2001047512 A2 WO 2000-US42661 20001206; AU 2001049041 A AU 2001-49041 20001206
FDT AU 2001049041 A Based on WO 2001047512
PRAI US 1999-457498 19991208
AB WO 200147512 A UPAB: 20010905
NOVELTY - Treatment of pain associated with an interior disease site in a subject, comprising administering at least one biologically compatible pain-relieving target construct to the subject, is new. The construct comprises a pain-relieving agent linked to a ligand moiety that selectively binds to or is taken up by the tissue associated with the painful interior disease site.

DETAILED DESCRIPTION - Treatment of pain associated with an interior disease site in a subject, comprising administering at least one biologically compatible pain-relieving target construct to the subject, is new. The construct comprises a pain-relieving agent linked to a ligand moiety that selectively binds to or is taken up by the tissue associated with the painful interior disease site. The construct is allowed to bind to and/or be taken up selectively by the tissue, thus delivering pain relief to the subject.

ACTIVITY - Analgesic.
No biological data is given.
MECHANISM OF ACTION - None given.
USE - For treating pain associated with an interior disease site.
ADVANTAGE - Since the pain-relieving agent is delivered by the ligand to the disease site intractable pain situated in the interior of the body such as caused by various tumors can be managed using a lower level of the pain relieving agent than is required when the pain-relieving agent is injected in the free state.
Dwg.0/0

L37 ANSWER 31 OF 42 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
AN 2001:219562 BIOSIS
DN PREV200100219562
TI Rho GTPases as modulators of the estrogen receptor transcriptional response.
AU Su, Laura F.; Knoblauch, Roland; Garabedian, Michael J. [Reprint author]
CS Dept. of Microbiology, NYU School of Medicine, 550 First Ave., New York, NY, 10016, USA
garabm01@med.nyu.edu
SO Journal of Biological Chemistry, (February 2, 2001) Vol. 276, No. 5, pp. 3231-3237. print.
CODEN: JBCHA3. ISSN: 0021-9258.
DT Article
LA English
ED Entered STN: 9 May 2001
Last Updated on STN: 18 Feb 2002
AB The estrogen receptor alpha (ER) is a ligand-dependent transcription factor that plays a critical role in the development and progression of **breast cancer**, in part, by regulating target genes involved in cellular proliferation. To identify novel components that affect the ER transcriptional response, we performed a genetic screen in yeast and identified RDI1, a Rho guanine nucleotide dissociation inhibitor (Rho GDI), as a positive regulator of ER transactivation. Overexpression

of the human homologue of RDI1, Rho GDIalpha, increases ERalpha, ERbeta, androgen receptor, and glucocorticoid receptor transcriptional activation in mammalian cells but not activation by the unrelated transcription factors serum response factor and Sp1. In contrast, expression of constitutively active forms of RhoA, Rac1, and Cdc42 decrease ER transcriptional activity, suggesting that Rho GDI increases ER transactivation by antagonizing Rho function. Inhibition of RhoA by expression of either the Clostridium **botulinum** C3 transferase or a dominant negative RhoA resulted in enhanced ER transcriptional activation, thus phenocopying the effect of Rho GDI expression on ER transactivation. Together, these findings establish the Rho GTPases as important modulators of ER transcriptional activation. Since Rho GTPases regulate actin polymerization, our findings suggest a link between the major regulators of cellular architecture and steroid receptor transcriptional response.

L37 ANSWER 32 OF 42 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2000:738879 HCAPLUS
DN 133:301197
TI Oxalic acid or oxalate compositions and methods for bacterial, viral, and other diseases or conditions
IN Hart, Francis J.
PA USA
SO U.S., 50 pp., Cont.-in-part of U. S. Ser. No. 629,538.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 6133318	A	20001017	US 1998-14943	19980128
	US 6133317	A	20001017	US 1996-629538	19960409
	US 6407141	B1	20020618	US 2000-535572	20000327
PRAI	US 1995-6785P	P	19951115		
	US 1996-629538	A2	19960409		
	US 1997-36983P	P	19970129		
	US 1998-14943	A2	19980128		

AB A single medicine oxalic acid or oxalate or "magic bullet" and method for treatment or prevention of infectious or pathogenic microbial, bacterial, viral and other diseases in warm-blooded animals, including humans and pets, is provided. A composition includes at least one therapeutically effective form of oxalic acid or oxalate selected from ester, lactone or salt form including sodium oxalate, oxalic acid dihydrate, anhydrous oxalic acid, oxamide, and oxalate salts, natural or processed foods including molds, plants or vegetables containing oxalic acid or oxalate, beverages, liqs. or juices containing oxalic acid or oxalate, additives containing oxalic acid or oxalate, and combinations thereof. The composition may also contain a pharmaceutically acceptable carrier or diluent for the therapeutically effective form of oxalic acid or oxalate. Methods are provided including the steps of periodically administering, by topical, oral, or parenteral application, a therapeutically effective dosage of a composition including at least one therapeutically effective form of oxalic acid or oxalate and improving chemotherapy reducing the intake of oxalic acid or oxalate blockers such as citric acid, ascorbic acid (vitamin C), pyridoxine hydrochloride (vitamin B6), calcium, alc., resins, clays, foods containing calcium, beverages containing alc., citric acid, or ascorbic acid, red meat or white meat of fowl containing pyridoxine hydrochloride, or other foods nutritional supplements or beverages containing oxalic acid or oxalate blockers.
RE.CNT 103 THERE ARE 103 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L37 ANSWER 33 OF 42 EMBASE
on STN
AN 2000067338 EMBASE
TI Tourette syndrome, associated conditions and the complexities of treatment.
AU Robertson M.M.
CS Prof. M.M. Robertson, Dept. Psychiat. Behavioural Sci., University College London, Wolfson Building, 48 Riding House Street, London W1N 8AA, United Kingdom. rejummr@ucl.ac.uk
SO Brain, (2000) 123/3 (425-462).
Refs: 476
ISSN: 0006-8950 CODEN: BRAIAK
CY United Kingdom
DT Journal; General Review
FS 008 Neurology and Neurosurgery
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English
AB Tourette syndrome (TS) is characterized by multiple motor tics plus one or more vocal (phonic) tics, which characteristically wax and wane. It can no longer be considered the rare and bizarre syndrome that it was once thought to be. The concepts surrounding TS, and our understanding of it, are also becoming increasingly complex and, in some individuals, TS is now recognized to be associated with a wide variety of associated behaviours and psychopathologies. It is suggested that TS is heterogeneous from a variety of standpoints including clinical presentation and psychopathology, and thus neuropharmacological responses and possibly even aetiological and genetic mechanisms. In this paper, mention is made of recent findings in epidemiology and genetics, highlighting the complexities of the disorder; these have been chosen because findings in both areas have clinical and management implications. The literature on the clinical manifestations, associated behaviours, psychopathology (and/or comorbid conditions) and management, in particular, is reviewed in detail.

L37 ANSWER 34 OF 42 MEDLINE on STN
AN 2001201496 MEDLINE
DN PubMed ID: 11191108
TI RhoC GTPase overexpression modulates induction of angiogenic factors in breast cells.
AU van Golen K L; Wu Z F; Qiao X T; Bao L; Merajver S D
CS Department of Internal Medicine, The University of Michigan Comprehensive Cancer Center, Ann Arbor 48109, USA.
NC 5T32 CA09537 - 16 (NCI)
R01 CA 77612 (NCI)
SO Neoplasia (New York, N.Y.), (2000 Sep-Oct) 2 (5) 418-25.
Journal code: 100886622. ISSN: 1522-8002.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200104
ED Entered STN: 20010417
Last Updated on STN: 20010417
Entered Medline: 20010412
AB Inflammatory breast cancer (IBC) is a distinct and aggressive form of locally advanced breast cancer. IBC is highly angiogenic, invasive, and

metastatic at its inception. Previously, we identified specific genetic alterations of IBC that contribute to this highly invasive phenotype. RhoC GTPase was overexpressed in 90% of archival IBC tumor samples, but not in stage-matched, non-IBC tumors. To study the role of RhoC GTPase in contributing to an IBC-like phenotype, we generated stable transfectants of human mammary epithelial cells overexpressing the RhoC gene, and studied the effect of RhoC GTPase overexpression on the modulation of angiogenesis in IBC. Levels of vascular endothelial growth factor (VEGF), basic fibroblast growth factor (bFGF), interleukin-6 (IL-6), and interleukin-8 (IL-8) were significantly higher in the conditioned media of the HME-RhoC transfectants than in the untransfected HME and HME-beta-galactosidase control media, similar to the SUM149 IBC cell line. Inhibition of RhoC function by introduction of C3 exotransferase decreased production of angiogenic factors by the HME-RhoC transfectants and the SUM149 IBC cell line, but did not affect the control cells. These data support the conclusion that overexpression of RhoC GTPase is specifically and directly implicated in the control of the production of angiogenic factors by IBC cells.

L37 ANSWER 35 OF 42 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN DUPLICATE 10
AN 1999-571873 [48] WPIDS
DNN N1999-421434 DNC C1999-166895
TI New heteromyeloma cell capable of producing trioma cell when fused with lymphoma cell, useful for treating cancer, autoimmune dysfunction, cardiovascular disease or transplantation.
DC A96 B04 D16 S03
IN TRAKHT, I
PA (UYCO) UNIV COLUMBIA NEW YORK
CYC 24
PI WO 9947929 A1 19990923 (199948)* EN 86
RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE
W: AU CA JP MX US
AU 9931889 A 19991011 (200008)
EP 1064551 A1 20010103 (200102) EN
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
US 6197582 B1 20010306 (200115)
JP 2002507398 W 20020312 (200220) 77
ADT WO 9947929 A1 WO 1999-US5828 19990318; AU 9931889 A AU 1999-31889 19990318; EP 1064551 A1 EP 1999-913925 19990318, WO 1999-US5828 19990318; US 6197582 B1 US 1998-40833 19980318; JP 2002507398 W WO 1999-US5828 19990318, JP 2000-537073 19990318
FDT AU 9931889 A Based on WO 9947929; EP 1064551 A1 Based on WO 9947929; JP 2002507398 W Based on WO 9947929
PRAI US 1998-40833 19980318
AB WO 9947929 A UPAB: 19991122
NOVELTY - A heteromyeloma cell which does not produce any antibody and is capable of producing a trioma cell which does not produce any antibody when fused with a human lymphoid cell is new.

DETAILED DESCRIPTION - A heteromyeloma cell (I) which does not produce any antibody and is capable of producing a trioma cell which does not produce any antibody when fused with a human lymphoid cell is new. The trioma cell is capable of producing a tetroma which produces a monoclonal antibody having a specific binding affinity for an antigen when fused with a second human lymphoid cell produces an antibody having a specific binding affinity for the antigen, with the proviso that the heteromyeloma cell is not B6B11 ATCC HB-12481.

INDEPENDENT CLAIMS are also included for the following:

(1) a trioma cell (II) which does not produce any antibody obtained by fusing a heteromyeloma cell which does not produce any antibody with a

human lymphoid cell;

(2) a tetroma cell (III) capable of producing a monoclonal antibody having a specific binding affinity for an antigen obtained by fusing (II) with a human lymphoid cell capable of producing an antibody having specific binding affinity for the antigen;

(3) a monoclonal antibody (IV) produced by (III);

(4) an isolated nucleic acid (V) encoding (IV);

(5) a method of generating (II) comprising:

(a) fusing a heteromyeloma cell which does not produce antibody with a human lymphoid cell therefore forming trioma cells;

(b) incubating the trioma cells formed in (a) under conditions permissive to the production of antibody by the trioma cells; and

(c) selecting a trioma cell that does not produce any antibody;

(6) a trioma cell generated by (5);

(7) a method for producing tetroma cells capable of producing a monoclonal antibody comprising:

(a) fusing the trioma cell with a human lymphoid cell therefore forming tetroma cells;

(b) incubating the tetroma cells formed in (a) under conditions permissive for the production of antibody by the tetroma cells; and

(c) selecting a tetroma cell capable of producing a monoclonal antibody;

(8) a tetroma cell generated by (7);

(9) a method for the production of a monoclonal antibody comprising:

(a) fusing a lymphoid cell capable of producing antibody with (II) to form tetroma cells; and

(b) incubating the tetroma cells formed in (b) under conditions permissive for the production of antibody by the tetroma cells, therefore producing the monoclonal antibody;

(10) a method of producing a monoclonal antibody specific for an antigen associated with a condition in a subject comprising:

(a) fusing a lymphoid cell capable of producing antibody with (II) to form tetroma cells;

(b) incubating the tetroma cells formed in (a) under conditions permissive for the production of antibody by the tetroma cells;

(c) selecting a tetroma cell producing a monoclonal antibody;

(d) contacting the monoclonal antibody of (c) with a sample from a subject with the condition or a sample from a subject without the condition under conditions permissive to the formation of a complex between the monoclonal antibody and the sample;

(e) detecting the complex formed between the monoclonal antibody and t he sample;

(f) determining the amount of complex formed in (e); and

(g) comparing the amount of complex determined in (f) for the sample from the subject with the condition with the amount determined in (f) for the sample from the subject without the condition, a greater amount of complex formation for the sample from the subject with the condition indicating that a monoclonal antibody specific for the antigen specific for the antigen specific for the condition produced;

(11) a monoclonal antibody produced by (9) and/or (10);

(12) a nucleic acid encoding the monoclonal antibody (11);

(13) a method for identifying an antigen associated with a condition in a sample, comprising:

(a) contacting the monoclonal antibody with the sample under conditions permissive to the formation of a complex between the monoclonal antibody and the sample;

(b) detecting the complex formed in (a); and

(c) isolating the complex detected in (b) therefore identifying the antigen associated with the condition in the sample;

(14) a tumor antigen identified by the method (12);

(15) a method for diagnosing a tumor in a sample comprising detecting the presence of the tumor antigen identified by the method, the presence of the antigen indicating the presence of tumor in the subject;

(16) a method for diagnosing a condition in a subject comprising:

(a) contacting a sample from the subject with the monoclonal antibody under conditions permissive to the formation of a complex between the monoclonal antibody and the sample; and

(b) detecting the complex formed between the monoclonal antibody and the sample, positive detection indicating the presence of an antigen specific for the condition in the sample, therefore diagnosing the condition in the sample;

(17) a composition comprising the monoclonal antibody and a suitable carrier; and

(18) a method for treating and preventing a condition in a subject comprising administering to the subject an amount of the therapeutic composition effective to bind the antigen associated with the condition, therefore treating the condition in the subject.

ACTIVITY - Cytostatic; Antibacterial; Antiviral; Immunosuppressive.

MECHANISM OF ACTION - The method is sufficient for inhibiting the growth of or the elimination of cancer (especially breast cancer, thyroid cancer or prostate cancer) and for inhibiting the growth of or for killing the infectious agent.

USE - The cells are useful for treating and preventing conditions e.g. Hanta virus, HTLV-I, HTLV II, HIV, herpes virus, influenza virus, Ebola virus, human papilloma virus, Staphylococcus, Streptococcus, Klebsiella, E. coli, anthrax or cryptococcus. The condition is associated with a toxin and the amount of monoclonal antibody is sufficient to reduce the amount or destroy the toxin (especially tetanus, anthrax, botulinum, snake venom or spider venom). The condition is an autoimmune disease (especially lupus, thyroiditis, graft versus host disease, transplantation rejection or rheumatoid arthritis). The condition is associated with a cancer (especially breast cancer, thyroid cancer or prostate cancer), a toxin, an infectious agent, an enzyme dysfunction, a hormone dysfunction, an autoimmune disease, an immune dysfunction, a viral antigen, a bacterial antigen, a eukaryotic antigen, or rejection of a transplanted tissue, septicemia, sepsis, septic shock, viremia, bacteremia or fungemia. The tumor is benign and the enzyme dysfunction is hyperactivity or overproduction of the enzyme. The hormone dysfunction is hyperactivity or overproduction of the hormone. The immune dysfunction is CD3 or CD4 mediated.

ADVANTAGE - No advantages specified in the specification.

Dwg.0/7

L37 ANSWER 36 OF 42 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 1999:709004 HCAPLUS
DN 131:321545
TI Methods of selecting internalizing antibodies
IN Marks, James D.; Poul, Marie-alix; Becerril, Baltazar
PA The Regents of the University of California, USA
SO PCT Int. Appl., 88 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9956129	A1	19991104	WO 1999-US8468	19990422

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK,

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            MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ,
            TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ,
            MD, RU, TJ, TM
        RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK,
            ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG,
            CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
     US 2001008759          A1    20010719    US 1999-249529          19990212
     US 6794128             B2    20040921
     CA 2326499             AA    19991104    CA 1999-2326499         19990422
     AU 9938622             A1    19991116    AU 1999-38622           19990422
     AU 768784              B2    20040108
     EP 1073905             A1    20010207    EP 1999-921396          19990422
        R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
           IE, FI
     JP 2002513156          T2    20020508    JP 2000-546239          19990422
     US 2005037339          A1    20050217    US 2004-855755          20040526
PRAI US 1998-82953P         P     19980424
     US 1999-249529         A     19990212
     WO 1999-US8468         W     19990422
AB   This invention provides methods of selecting antibodies that are
     internalized into target cells. The methods generally involve contacting
     target cells with one or more members of an antibody phage display
     library, shown in the figure. The members of the phage display library
     are also contacted with cells of subtractive cell line. The target cells
     are then washed to remove the subtractive cell line cells and members of
     phage display library that are non-specifically bound or weakly bound to
     the target cells. The target cells are cultured under conditions where
     members of the phage display library can be internalized if bound to an
     internalizing marker and internalized members of the phage display library
     are then identified.
RE.CNT 3     THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
             ALL CITATIONS AVAILABLE IN THE RE FORMAT

L37  ANSWER 37 OF 42  HCAPLUS  COPYRIGHT 2005 ACS on STN
AN   1999:529160  HCAPLUS
DN   131:165335
TI   Sphingolipid derivatives, their preparation, and their therapeutic use
IN   Liotta, Dennis C.; Merrill, Alfred H., Jr.; Keane, Thomas E.; Schmelz, Eva
     M.; Bhalla, Kapil N.
PA   Emory University, USA
SO   PCT Int. Appl., 140 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.          KIND   DATE        APPLICATION NO.         DATE
     --------------      ----   --------    --------------------    --------
PI   WO 9941266           A1    19990819    WO 1999-US3093          19990212
        W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE,
           ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS,
           LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD,
           SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, AM, AZ, BY,
           KG, KZ, MD, RU, TJ, TM
        RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,
           PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
     CA 2320117             AA    19990819    CA 1999-2320117         19990212
     AU 9927644             A1    19990830    AU 1999-27644           19990212
     AU 765809              B2    20031002
     EP 1053243             A1    20001122    EP 1999-908143          19990212
        R: DE, FR, GB, IT, IE
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	US 6610835	B1	20030826	US 1999-249211	19990212
	US 2004039212	A1	20040226	US 2003-647801	20030825
PRAI	US 1998-74536P	P	19980212		
	US 1999-249211	A1	19990212		
	WO 1999-US3093	W	19990212		

OS MARPAT 131:165335

AB Derivs. of sphingolipids (Markush included) are provided. The compds. are useful in the treatment of abnormal cell proliferation, including benign and malignant tumors, the promotion of cell differentiation, the induction of apoptosis, the inhibition of protein kinase C, and the treatment of inflammatory conditions, psoriasis, inflammatory bowel disease as well as proliferation of smooth muscle cells in the course of development of plaques in vascular tissue. The invention also includes a method for triggering the release of cytochrome c from mitochondria that includes administering an effective amount of a sphingolipid or its derivative or prodrug to a host in need thereof. Further, the invention provides a method for treating bacterial infections, including those that influence colon cancer and other disorders of the intestine, that includes administering an effective amount of one of the active compds. identified herein.

RE.CNT 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L37 ANSWER 38 OF 42 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

AN 2000:197816 BIOSIS

DN PREV200000197816

TI Clinical phase II evaluation of the combination therapy with docetaxel and epidoxorubicin in the neoadjuvant, cytostatic treatment on patients with primary **breast cancer** (T1-4, N0-2, M0).

AU Wenzel, Catharina; Schmidinger, Manuela; Locker, Gottfried J.; Taucher, Susanne; Gnant, Michael; Jakesz, Raimund; Steger, Guenther G. [Reprint author]

CS Klinische Abteilung fuer Onkologie, Universitaetsklinik fuer Innere Medizin I, Waehringer Guertel 18-20, A-1090, Wien, Austria

SO Wiener Klinische Wochenschrift, (Oct. 29, 1999) Vol. 111, No. 20, pp. 843-850. print.
CODEN: WKWOAO. ISSN: 0043-5325.

DT Article

LA German

ED Entered STN: 17 May 2000
Last Updated on STN: 4 Jan 2002

AB Background: Preoperative (neo-adjuvant) chemotherapy is very effective in downstaging primary tumors and moreover is able to prevent advancing metastatic growth early in the course of the disease. Methods: We report on 38 patients with a median age of 54 years (range, 33-70 years) suffering from biopsy-proven **breast cancer** (T1-T4). Mastectomy had been considered the treatment of choice in all cases. The patients received 194 cycles of chemotherapy with docetaxel (75 mg/m2) and epidoxorubicin (75 mg/m2) on day 1, every 21 days, together with 30 million IU of G-CSF from days 3 to 10. Three to 8 cycles (median 5 cycles) of the treatment were administered until best response was achieved on mammography and clinical assessment. Results: The neo-adjuvant chemotherapy was well tolerated and all patients completed the treatment regimen on an out-patient basis. During 194 cycles we observed leukopenia WHO grade IV only at one occasion (0.5%). WHO-grade III toxicity consisted of leukopenia (0.5%), diarrhoea (2%), and stomatitis (0,5%). Response to treatment was present in 85%, with 4 patients (11%) experiencing a pathological complete response (pCR) of the invasive tumor (T0: n = 2, DCIS: n = 2) and 28 patients (74%) showing a

partial pathological response. In 21 patients (52%) a breast-conserving surgical procedure was possible. Summary: We conclude that neo-adjuvant treatment of primary **breast cancer** with docetaxel and epidoxorubicin is safe and effective. By applying more chemotherapy cycles preoperatively it might even be possible to raise the rate of pCR and prolong survival.

L37 ANSWER 39 OF 42 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN DUPLICATE 11
AN 1999274285 EMBASE
TI Rho-kinase (ROK) promotes CD44v3,8-10-ankyrin interaction and tumor cell migration in metastatic breast cancer cells.
AU Bourguignon L.Y.W.; Zhu H.; Shao L.; Zhu D.; Chen Y.-W.
CS Dr. L.Y.W. Bourguignon, Department of Cell Biology/Anatomy, University of Miami Medical School, 1600 N.W. 10th Avenue, Miami, FL 33136, United States. lbourgui@mednet.med.miami.edu
SO Cell Motility and the Cytoskeleton, (1999) 43/4 (269-287).
Refs: 75
ISSN: 0886-1544 CODEN: CMCYEO
CY United States
DT Journal; Article
FS 016 Cancer
029 Clinical Biochemistry
LA English
SL English
AB Metastatic breast tumor Met-1 cells express CD44v3,8-10, a major adhesion receptor that binds extracellular matrix components at its extracellular domain and interacts with the cytoskeletal protein, ankyrin, at its cytoplasmic domain. In this study, we have determined that CD44v3,8- 10 and RhoA GTPases are physically associated in vivo, and that CD44v3,8-10-bound RhoA displays GTPase activity, which can be inhibited by botulinum toxin C3-mediated ADP-ribosylation. In addition, we have identified a 160 kDa Rho-Kinase (ROK) as one of the downstream targets for CD44v3,8-10-bound RhoA GTPase. Specifically, RhoA (complexed with CD44v3,8-10) stimulates ROK-mediated phosphorylation of certain cellular proteins including the cytoplasmic domain of CD44v3,8-10. Most importantly, phosphorylation of CD44v3,8-10 by ROK enhances its interaction with the cytoskeletal protein, ankyrin. We have also constructed two ROK cDNA constructs that encode for proteins consisting of 537 amino acids [designated as the constitutively active form of ROK containing the catalytic domain (CAT, also the kinase domain)], and 173 amino acids [designated as the dominant-negative form of ROK containing the Rho-binding domain (RB)]. Microinjection of the ROK's CAT domain into Met-1 cells promotes CD44-ankyrin associated membrane ruffling and projections. This membrane motility can be blocked by CD44 antibodies and cytochalasin D (a microfilament inhibitor). Furthermore, overexpression of a dominant-negative form of ROK by transfection of Met-1 cells with ROK's Rho-binding (RB) domain cDNA effectively inhibits CD44-ankyrin-mediated metastatic behavior (e.g., membrane motility and tumor cell migration). These findings support the hypothesis that ROK plays a pivotal role in CD44v3,8-10-ankyrin interaction and RhoA-mediated oncogenic signaling required for membrane- cytoskeleton function and metastatic tumor cell migration.

L37 ANSWER 40 OF 42 MEDLINE on STN DUPLICATE 12
AN 1999196933 MEDLINE
DN PubMed ID: 10094832
TI Activation of protein kinase C by phorbol esters modulates alpha2beta1 integrin on MCF-7 breast cancer cells.
AU Rosfjord E C; Maemura M; Johnson M D; Torri J A; Akiyama S K; Woods V L

Jr; Dickson R B
CS Lombardi Cancer Research Center, Georgetown University, Washington, DC, 20007, USA.
NC 2P30-CA-51008 (NCI)
2P50-CA58185-04 (NCI)
IP50CA58185 (NCI)
SO Experimental cell research, (1999 Apr 10) 248 (1) 260-71.
Journal code: 0373226. ISSN: 0014-4827.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199906
ED Entered STN: 19990614
Last Updated on STN: 19990614
Entered Medline: 19990603
AB Cellular adhesions to other cells and to the extracellular matrix play crucial roles in the malignant progression of cancer. In this study, we investigated the role of protein kinase C (PKC) in the regulation of cell-substratum adhesion by the breast adenocarcinoma cell line MCF-7. A PKC activator, 12-O-tetradecanoylphorbol-1, 3-acetate (TPA), stimulated cell adhesion to laminin and collagen I in a dose-dependent manner over a 1- to 4-h interval. This enhanced adhesion was mediated by alpha2beta1 integrin, since both anti-alpha2 and anti-beta1 blocking antibodies each completely abrogated the TPA-induced adhesion. FACS analysis determined that TPA treatment does not change the cell surface expression of alpha2beta1 integrin over a 4-h time interval. However, alpha2beta1 levels were increased after 24 h of TPA treatment. Thus, the enhanced avidity of alpha2beta1-dependent cellular adhesion preceded the induction of alpha2beta1 cell surface expression. Northern blot analysis revealed that mRNA levels of both alpha2 and beta1 subunits were increased after exposure to TPA for 4 h, indicating that the induction of alpha2beta1 mRNA preceded that of its cell surface expression. This further suggested that the TPA-induced avidity of alpha2beta1 was independent of increased expression of alpha2beta1. Pretreatment of cells with the PKC inhibitor calphostin C partially antagonized the TPA-induced increase in expression of alpha2beta1 integrin expression and of alpha2beta1-mediated cellular adhesion. To identify a possible mechanism by which TPA could be acting to promote the rapid induction of alpha2beta1 adhesion, we treated the cells with the Rho-GTPase inhibitor Clostridium botulinumexotoxin C3. C3 inhibited TPA-induced adhesion to laminin and collagen I in a dose-dependant manner, suggesting a likely role for Rho in TPA-induced adhesion. Together, these results suggest that PKC can modulate the alpha2beta1-dependent adhesion of MCF-7 cells by two distinct mechanisms: altering the gene expression of integrins alpha2 and beta1 and altering the avidity of the alpha2beta1 integrin by a Rho-dependant mechanism.

L37 ANSWER 41 OF 42 MEDLINE on STN
AN 1998112733 MEDLINE
DN PubMed ID: 9452354
TI Neuromyotonia in a muscle flap producing a convulsing breast: successful treatment with botulinum toxin.
AU Schwartz M S; Wren D R; Filshie J
CS Atkinson Morleys Hospital, Wimbledon, England.
SO Movement disorders : official journal of the Movement Disorder Society, (1998 Jan) 13 (1) 188-90.
Journal code: 8610688. ISSN: 0885-3185.
CY United States
DT (CASE REPORTS)

Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199803
ED Entered STN: 19980407
Last Updated on STN: 19980407
Entered Medline: 19980326

L37 ANSWER 42 OF 42 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 94135818 EMBASE
DN 1994135818
TI [Drug market 1993. What was really new? Part 4].
ARZNEIMITTELMARKT 1993. WAS WAR WIRKLICH NEU? - TEIL 4.
AU Fricke U.
CS Institut fur Pharmakologie, Universitat zu Koln, Gleueler Strasse 24,50867 Koln, Germany
SO Deutsche Apotheker Zeitung, (1994) 134/17 (23-36).
ISSN: 0011-9857 CODEN: DAZEA2
CY Germany
DT Journal; General Review
FS 008 Neurology and Neurosurgery
012 Ophthalmology
016 Cancer
024 Anesthesiology
030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LA German

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